EXHIBIT 27

RESPONSE OF DR. FRANK AND DR. WHITEHOUSE TO REPORT OF THE ACC'S DR. L. WELCH March 2009.

1. Primary cause of death.

To make the CARD mortality study more comparable to the insulators studies, deaths from non-malignant asbestos-related disease (ARD) were evaluated in terms of primary cause and death certificates were re-evaluated in terms of underlying cause. See spreadsheet "CARD Mortality Study Non-Malignant ARD Deaths Primary/Contributing Cause." Exh. 7 to Whitehouse Report May 2009. Also, 13 death certificates had ARD only as a contributing factor, and were not used in the comparison to the insulators studies.

2. Hospital records.

Since all subjects in the cohort were CARD or Dr. Whitehouse patients, as treating physicians we often had important information in excess of that available to Selikoff et al. Conservatively, where we did not have all hospital records on the deceased and where there was uncertainty as to whether ARD was a primary cause or significant contributing factor in the death to a medical probability, the death was not called out as an ARD death. LB, WC, and SB, were all cases CARD was familiar with and determinations of cause of death were reasonable.

3. "Eight pleural deaths."

Welch, p.6, somehow selects eight pleural deaths. No explanation is given for how she selected them. The Whitehouse Report 12/29/08, ¶ 31(3), finds 27 with no interstitial disease on last chest x-ray. Eight had CT scans, but Dr. Whitehouse did not select the eight with CT scans as "eight pleural deaths." At this point, more CT scans have been read than back in December 2008, and information is restated at Whitehouse Report May 2009, ¶ 31(3). We note that Dr. Welch disagrees with some of Dr. Whitehouse's determinations on cause of death.

4. Whitehouse (2004).

The paper is titled "Asbestos Related Pleural Disease Due to Tremolite Associated With Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana," Am J Ind Med 46:219-225 (2004).

For this section we will follow the numbering in the Welch Report March 2009, pages 7-12.

(1) Patients diagnosed are at risk.

Whitehouse (2004), p.224, states:

> It is apparent from these data that the majority of the 1,500 persons who have radiologic changes of asbestos exposure are at increased risk for progressive loss of lung function from pleural changes alone or from potential future development of interstitial fibrosis.

Welch, p.7, writes: "It is my opinion that one cannot extrapolate from this group of 123 patients to the larger cohort of 1,500 people in Libby, Montana, with radiologic changes due to asbestos exposure." This opinion is based solely on differences in exposure as between miners and community members. Clinical observation of the cohort of CARD patients over the last eight years clearly indicates that those diagnosed are "at risk for progressive loss of lung function." That is a very conservative statement. Many, many patients progress, whether their exposure has been heavy or relatively light. It appears that once the disease process takes hold, it is generally progressive regardless of exposure level.

(2) Asbestos disease population.

Since at least 2007, Dr. Whitehouse has clarified that the 123 subjects in the Whitehouse (2004) study are representative of the Libby area asbestos disease population. No statistical extensions have been made to the "Libby residents."

(3) Height.

Dr. Welch does not give any numbers for height loss over a three year

period. Based upon decades of clinical practice, following patients for long periods of time with serial PFTs, Dr. Whitehouse observes that such height loss is negligible and would have no significant effect on PFT results.

(4) Lung function decline in current smokers.

Welch, p.8, acknowledges that only 7% of subjects in the Whitehouse (2004) study were current smokers. This is too small a number to make a significant difference. Both asbestos disease and smoking may cause obstructive disease.

(5) Cross sectional data.

Dr. Welch presents a point of academic interest, which has no relation to the real world. Welch, p.8, n.4, suggests studying "the same individual at age 40 and then 20 years later at age 60." All of the longitudinal lung function studies on asbestos disease patients are "cross-sectional." Apparently Dr. Welch disagrees with them all and would have them all tossed out. For example see Jones et al (1989), Ohlson et al (1985), Siricusa et al (1984), Murphy et al (1971, 1978), Epler et al (1979), Rom et al (1992), Whitehouse (2004), and Alfonso et al (2005).

(6) Predicted values.

Welch, p.8, writes:

> The predicted values used in Dr. Whitehouse's analysis actually have few data points in their age range of his population, making the "expected" loss of lung function with age subject to a wide confidence interval.

In fact, the studies used to established predicted values include thousands of participants. There is no wide confidence interval.

(7) Body mass index.

Whitehouse (2004), p.221, states:

Over the course of the study group observation, average BMI

increased less than 1 kg/m2 and there was no statistical correlation between increasing BMI and loss of lung function.

Welch seems to disagree with the statistics without doing her own.

(8) Interstitial disease was minimal.

Welch, p.9, writes: "There actually were only 67 patients with pleural disease and no interstitial disease." Welch assumes that all 56 patients with 0/1 or 1/0 for interstitial changes on chest x-ray actually had "interstitial disease." ATS (2004) Official Statement, p.700, states at "the level of 1/0 is used as the boundary between normal and abnormal in the evaluation of the film." Dr. Welch cannot assume that any of the 0/1 films, were abnormal.

(9) Pulmonary function controls.

All standards were met.

(a) Equipment changes.

All standards were met.

In clinical practice, pulmonary function tests (PFTs) done on different equipment at different locations are relied upon, per ATS standards. One keeps in mind that different locations may produce different results. However, for quite some time now all labs are computerized, which has resolved many control issues. Three of the four labs mentioned by Dr. Welch were supervised by Dr. Whitehouse.

(b) Minimize biological variability.

All standards were met.

At pages 10-11, Welch apparently quotes without citation the ACOEM Position Statement on Spirometry in the Occupational Setting (2000). ACOEM, p.238, states in similar fashion:

In subjects with "normal" lung function, changes in FVC or FEV1

> over one year should probably exceed 15% before any confidence can be given to the opinion that a meaningful year-to-year change has occurred.

Both the quote and context of the quote relate to individual subjects. Indeed, Dr. Welch's quotes at page 11 specifically relate to individual subjects. In a cohort of 123 subjects, individual variations sort themselves out statistically. This is elementary. Thus ATS (2004) Official Statement, p.705, considers loss of "about 5% of FVC" over time as significant. See also Ohlson et al (1985), where a cohort with a change in FVC 7% and FEV1 6% over four years was considered significant. See Alfonso et al (2005), p.186, where a cohort with a 2.2% per year decline in DLCO was considered significant.

Welch considers changes under 15% per year over time as clinically insignificant. This is reckless. Welch would consider a 14% per year change as clinically insignificant. A lung function loss of 14% per year for three years is a 42% loss of volume. The patient could go from mild to severe without being noticed by Dr. Welch.

(10) DLCO has low variability.

Welch, p.11, writes: "The ATS statement on DLCO cites a 9% variation in normal individuals over a one year period." The cite is ATS/ERS (2005) DLCO, p.728. The study cited for the 9% variability is dated 1989. Modern computerized equipment generally does not have the problem of high variability, and DLCO is often more repeatable than is TLC.

Welch, p.11, states that the "American Thoracic Society recommends specific adjustment for hemoglobin, COHb, and inspired pressure of oxygen (altitude)." ATS (1995), "Single Breath Carbon Monoxide Diffusing Capacity," p.2194, states: "All current methods of adjusting for hemoglobin involve unproven assumptions, and no method has been uniformly accepted." Adjustment for hemoglobin is certainly not standard practice in the area of Spokane, Washington. It is rather rare that the adjusted number is different. It takes significant anemia to modify the DLCO number. Unless hemoglobin is very low, it does not make any difference in diffusion capacity (DLCO). Dr. Welch offers no amount for effect of a hemoglobin adjustment. Altitude is considered when selecting reference norms.

Welch, p.12, writes that there are "large inter-lab differences in measured DLCO," referring to ATS/ERS (2005) Interpretative Strategies, which cites 1993 and 1995 studies. In the real world, with modern computerized equipment, inter-lab differences are much less problematical. As Dr. Welch has acknowledged, DLCO is a routine measure of lung function in clinical practice, recommended by ATS (2004) Official Statement, and required by the AMA Guides for the Evaluation of Permanent Impairment.

The 15% variation in DLCO is for individual subjects over one year. Dr. Welch does not understand that a loss of 2.2% in diffusion capacity per year in an entire cohort is significant. See Alfonso et al (2005).

(11) Smoking and obstructive disease.

Welch, p.12, writes:

> There are a range of other factors which suggests his patients would have more rapid loss of lung function than the average healthy population, since he included some patients who were current and ex-smokers, patients who had respiratory symptoms, and patients being treated with bronchodilators presumably for some obstructive lung disease.

First, an "average population" includes current and ex-smokers. The Kundson norms were done on an average population which included smokers.

Smoking causes obstructive disease. Asbestos causes obstructive disease. Whitehouse (2004), p.220, excludes patients with "chronic obstructive pulmonary disease with elevated residual volumes." The ACC and Dr. Welch have had the study data since at least 2008, but did no analysis of their own.

As to the inclusion of "patients with respiratory symptoms," of course they are included! The paper concerns "progressive loss of lung function." If those with respiratory symptoms were excluded, there would be no study. This is a strange comment by Dr. Welch.

As to "patients being treated with bronchodilators presumably for

some obstructive lung disease," Dr. Welch "presumes," whereas Dr. Whitehouse knows the patients. Whitehouse (2004) states that Dr. Whitehouse had not entered a "diagnosis of bronchial asthma" on any of the 123 patients in the study.

(12) Individuals with parenchymal asbestosis.

Dr. Welch writes:

> Since almost half of Dr. Whitehouse's patient population had parenchymal asbestosis, one would not be surprised to see lung function loss in excess of what is due to aging in a healthy population.

This is incorrect. Whitehouse (2004), p.221, states: "The remaining patients (56) [45%] had minimal radiographic evidence of irregular interstitial changes involving the bases at refusion category 0/1 or 1/0." ATS (2004) Official Statement, p.700, states: "A profusion of irregular opacities at the level of 1/0 is used as the boundary between normal and abnormal." Dr. Welch leaps to the conclusion that all 0/1 films be diagnosed as "parenchymal asbestosis." This is an error. One may not assume that "almost half of Dr. Whitehouse's patient population had parenchymal asbestosis."

Dr. Welch misses an important point in the article. The 55% of patients with pleural disease only had the same lung function loss as the whole cohort. Compare Whitehouse (2004), Fig. 4 and Fig. 1. This indicates that the patients with minimal interstitial changes did not make an excess contribution to lung function loss.

5. Rapid radiographic progression.

Welch, p.12, misreads the Whitehouse Report 12/29/08. She states:

> Dr. Whitehouse identified 18 individuals as having rapid progression of pulmonary function loss.

The Whitehouse Report 12/29/08, ¶ 40, refers to radiographic progression, not progression of pulmonary function loss, and attaches at Exhibit 6 a CD

with films demonstrating radiographic progression. Information on lung function progression is attached at Exhibit 6 as well.

There are problems with Dr. Welch's work. Her report at pages 12-13, mentions various individuals. We obtained from the ACC the names of the individuals referenced. Mary Gerr, Ron Masters, and CC are not on the Exhibit 6 list of 18 individuals with rapid radiographic progression. Dr. Welch apparently did not use the list attached as Exhibit 6 to the Whitehouse Report of 12/29/08.

Welch claims to have reviewed the records and states that HC has obstructive disease. HC is a non-client, and no medical records for non-clients have been delivered.

A series of films is attached on a CD, as described on the exhibit list for Exhibit 6, Whitehouse Report 12/29/08. Dr. Welch does not comment on the films, except to say that "two of the individuals had diffuse pleural thickening."

Dr. Welch comments on obstructive disease without indicating its significance. Welch does not apparently disagree with ATS (2004), Official Statement, p.708, which states: "Asbestos exposure has long been known to be associated with an obstructive physiologic or abnormality." Having reviewed the medical records, Dr. Welch does not find that any of the 13 have emphysema or other smoking disease. Robert Mack and Wendy Challinor do not have an obstructive defect. Al Dickerman did not have obstructive disease, and never smoked. Jack DeShazer had elevated residual volumes, but had no smoking related disease.

Where DLCO tests do not meet all requirements, the interpreting physician must use judgment as "tests that are less than optimal may still contain useful information." ATS/ERS (2005), "Interpretative Strategies," p.948.

6. Studies on radiographic progression of asbestos disease.

Dr. Welch, p.15, writes that Dr. Frank and Dr. Whitehouse's chart at Whitehouse Report Exh. 13 "simplifies the studies." In response, Dr. Welch produces a chart which complicates the studies, to a point that Dr. Welch

sees no trend. With all the usual problems of comparing medical studies, the trend is still apparent. Amphibole disease is significantly more radiographically progressive.

Dr. Welch, p.15, states that the minimum latency for asbestosis is 10 years. Then Welch suggests that the Viallat (1983) and Shepherd (1997) studies be included. Viallat (1983) had a median latency to first x-ray of 13 years, meaning that in half of the cases latency was less than 13 years, which is significantly less than the 20 year expected latency. One would not expect abnormals on a first x-ray. Likewise, Shepherd (1997) had a median latency to first x-ray of 11 years, and the same problem.

7. The TDP requires blunting for diffuse pleural thickening.

Dr. Welch, p.18, offers the opinion that the use of ILO classifications to measure "severity of pulmonary function impairment" is medically reasonable. Welch cites two studies on interstitial fibrosis, but none on pleural disease.

Welch, p.18, writes: "There is strong scientific rationale for requiring blunting of the costophrenic angle in determining which pleural scarring causes significant impairment. Ameille et al (2004), at p.294, proposes that obliteration of the costophrenic angle be a criterion for diagnosis of DPT. As we have noted before, neither the ATS (2004) Official Statement, nor lung disease texts add on obliteration of the costophrenic angle (blunting) to the definition of diffuse pleural thickening. Welch, p.18, acknowledges that the ILO "classification was not designed for clinical diagnosis." Ameille et al (2004) cites three studies showing a correlation between blunting and decreases in FVC, FEV1, and TLC. These are Schwartz et al (1990), Lilis et al (1991), and Bourbeau et al (1990). All three studies arbitrarily define DPT as requiring blunting. Then comparisons were made with pleural plaques on loss of lung function. Not surprisingly comparing loss of lung function in patients with DPT and blunting exceeds that in patients with mainly pleural plaques. This is a comparison of apples and oranges. More interesting would be to compare loss of lung function in patients with DPT and blunting, as against patients with DPT per width and extent criteria.

There was no comparison of loss of lung function for DPT with and without blunting in the three studies above. Thus they provide no medical

basis for excluding patients with DPT and no blunting from the TDP medical criteria's definition of diffuse pleural thickening.

McLoud et al (1985), Fig. 10, did compare lung function loss for three types of diffuse pleural thickening, (1) pleural thickening due to "confluent plaques," (2) pleural thickening "post effusion," and (3) pleural thickening due to "asbestosis" ("extension of pulmonary fibrosis to visceral and parietal pleura," p.12). The "post effusion" category can represent a blunting category since 90% of the "post effusion" cases had blunting, and these cases represented 93% of the cases of blunting (52 of 56). The results at Fig. 10 show that the three causes of pleural thickening were quite consistent in lung function loss over increasing degrees of asbestosis. "Post effusion" cases of pleural thickening incurred somewhat more loss of vital capacity, but the difference surely could not justify tossing out all cases of pleural thickening due to "confluent plaques" and/or "asbestosis" from the definition of pleural thickening. A similar pattern was found on Fig. 10 for loss of diffusion capacity (DLCO) for all three forms of diffuse pleural thickening.

The Ameille et al (2004) data at Table 4 indicate that if the definition of diffuse pleural thickening is limited to patients with blunting, then patients with DPT along over 25% of the chest wall, but no blunting, end up in the "pleural plaques" group. Then the pleural plaques group is tossed out. This "pleural plaques" group containing many true DPT cases has 35% with exertional dyspnea and 19% with chest pain.

Interestingly, Bourbeau et al (1990), p.840, made some differentiation by "width and extent" of pleural thickening and stated:

> Although not shown in the Table, as for any pleural abnormality, complaint for dyspnea with major activities was statistically significant ($p<0.05$) when related to chest wall pleural plaques taken as a continuous variable according to width and extent independently of parenchymal disease.

Table 3 shows an odds ratio of 4.5 for "chest wall" pleural thickening, as compared to 2.1 for patients with blunting of the costophrenic angle.

Lee et al (2003) was a study of Wittenoom asbestos miners with

amphibole exposure. The authors developed a DPT score, based upon width and extent of pleural thickening. Presence or absence of blunting was noted, but did not enter into the scoring. The authors found that the "pleural thickening score correlated inversely with FEV1, FVC and TLC" (p.203). Lee et al (2003), Table 2, shows slightly lower values for FVC and TLC than does the Ameille Table 4 "DPT group." So, it does appear that there may be a similar lung function loss correlation whether the variable be blunting or width and extent of pleural thickening. And, Lee et al (2003) as an amphibole study is more relevant to Libby issues than the other three studies discussed above.

McLoud et al (1985) presents the best data on types of diffuse pleural thickening. The abstract states:

> Diffuse pleural thickening was defined as a smooth, non-interrupted pleural density extending over at least one-fourth of the chest wall, with or without costophrenic angle obliteration.

This is consistent with the standard definition for diffuse pleural thickening in ATS (2004) and various text books. McLoud et al (1985) carefully examined films for 185 patients and sorted them as follows, at Table 3:

Cause of Diffuse Thickening	No.	Percentage
Benign asbestos diffusion	58	31%
Confluent plaques	47	25%
Extension of pulmonary fibrosis (asbestosis)	19	10%
Malignancy, infection or trauma	47	25%
Obesity	5	3%
Unexplained	9	5%
Total	185	

McLoud et al (1985), p.12, apparently reports only 56 of 185 cases with

blunting, or 30%. Adopting medical criteria which only recognize diffuse pleural thickening with blunting could exclude 68 cases of true diffuse pleural thickening in the first three categories in the table above (58 + 47 + 19 = 124 - 56 = 68).

Ameille et al (2004), p.294, recognizes the findings of McLoud et al (1985) that diffuse pleural thickening may be due to (1) effusions, (2) confluence of plaques, or (3) extension of pulmonary fibrosis into the pleura. McLoud et al (1985) is frequently cited on this issue, and is followed in medical texts. ATS (2004) references McLoud et al (1985) and the three types of DPT at p.707.

Ameille et al (2004), p.294, next states:

> However, there is now a large consensus that DPT is the radiographic expression of thickening and fibrosis of the visual pleura, often associated with fusion of the parietal pleura, following clearance of benign asbestos-related pleural effusion. [Hillerdahl et al., 1990; Schwartz, 1991; Solomon, 1991; Miller et al., 1993; Rudd 1996; Consensus Report, 1997; Gevenois et al., 1998; Chailleux and Letourneux, 1999; Peacock et al., 2000].

Ameille et al (2004) imply that DPT <u>always</u> follows pleural effusion and should be defined as such. (Indeed DPT must <u>always</u> be due to effusion to support the author's conclusion.)

In fact, McLoud et al (1985), p.16, states:

> We have reported previously 35 cases of benign effusion among the same survey group of 1,135 asbestos exposed workers studied in this report, a prevalence of 3.1% [8]. Residual diffuse thickening, usually with a blunted angle was noted in half of those cases (54.3%).

We did not find that any of the articles cited by Ameille above states that DPT only follows "clearance of benign asbestos-related pleural effusion." (We note that Solomon (1991) was not available, and two other

articles were in French). The Ameille unsupported observation is certainly contrary to the detailed large study done by McLoud et al (1985) which documents in detail that only a minority of DPT cases result from pleural effusion. Ameille et al could have analyzed their own data for evidence of effusions.

Ameille et al (2004), p.294, next states:

> Several studies have confirmed that DPT is preceded by a benign asbestos pleural effusion [Martensson et al., 1987; Miller and Miller, 1993].

In Miller and Miller (1993), six cases were considered. In five cases DPT was preceded by pleural effusion. One may not generalize on a sample of six cases in any event. In Martensson et al (1987), there is no mention of DPT following effusion to any large degree. Apparently only seven of 71 patients with effusions had DPT.

Ameille et al (2004), p.294, concludes:

> Obliteration of the costophrenic angle represents the sequella of pleural effusion. It therefore appears logical to propose this criterion for the diagnosis of DPT.

There is no evidence in the studies cited that DPT always results from pleural effusion. Likewise there is no evidence in the studies cited that pleural effusions always cause blunting. In McLoud et al (1985), 90% of effusions led to blunting. McLoud cites Epler et al (1985) where 54% of asbestos pleural effusions led to blunting. Blunting often accompanies DPT. However, per McLoud et al (1985), a requirement of blunting before DPT is diagnosed would exclude a majority of cases of true DPT, which do not happen to have associated blunting of the costophrenic angle, but have DPT along a significant portion of the chest wall.

Ameille et al (2004) was not a well designed study. The authors had an opportunity to use their data to determine how well chest x-rays determine true DPT and how well the suggested definitions determine true DPT. Ameille et al (2004), p.290, states that "HRCT (high resolution CT

scan) was considered to be the gold standard for the diagnosis of pleural thickening and pulmonary fibrosis." The authors had 287 subjects with "pleural thickening" on CT scans. How many of the 287 had only pleural plaques? How many of the 287 had pleural thickening on CT with or without blunting (DPT per ATS (2004))? How many of these had DPT per definition one (blunting), or definition two (extent > 25% and 5mm thickness)? We are not told. It would have been best to present the data on the 287 CTs in detail so that one could examine the distribution of data.

Probably a majority of subjects on HRCT had pleural plaques only and no symptoms. Lilis et al (1992), p.50, notes that pleural plaques represent "in most exposed populations, about 80% of all pleural fibrosis." It would have been enlightening to be able to exclude the plaques only cases, and examine how well the chest x-ray definitions of DPT operated on cases confirmed by HRCT as DPT.

In the last two paragraphs of the article, the authors pick definition one (blunting) as a "much more reliable sign" of DPT in the visceral pleura than definition two (width and extent). The authors announce without presenting data that the dimensional criteria (width and extent) "cannot reliably extinguish between fibrosis of visceral and parietal pleura." If this is the key distinction, then there must be some data to support it. In practice, when reading chest x-rays and CT scans one has the same difficulty discerning the visceral pleura from the parietal pleura regardless of the definition of DPT.

Ameille et al (2004), pp.294-295, also announce that dimensional criteria (width and extent) "cannot reliably distinguish . . . between pleural fibrosis and subpleural fat pads." Again, no data is presented in support of the assertion. Again, in practice when reading chest x-rays and CT scans one has the same difficulties discerning the difference between pleural fibrosis and subpleural fat pads, regardless of the definition of DPT. Any difficulty in discerning pleural fibrosis from fat pads does not argue for a definition of a DPT that requires blunting. Blunting has nothing to do with it. The authors may have had the data to answer questions of discernment of true DPT per HRCT reading, versus subpleural fat pads. Discernment is easier on HRCT. But, the authors did not present the data.

Effusions occur without resulting in blunting. In Epler et al (1985) blunting is seen in the absence of any evidence of effusion in 46% of cases.

DPT is often seen without blunting, McLoud et al (1985). DPT is usually seen without evidence of effusion. Where DPT is seen over 25% of the chest wall and over 3mm in thickness, with or without blunting, it should be called diffuse pleural thickening. It should not be called a plaque, where it is diffuse, non-circumscribed, and blended on the edges. Where there is no blunting, the ILO system calls diffuse pleural thickening a "pleural plaque." This is a false result.

Dr. Welch, p.19, acknowledges that "there may be individuals who have significant diffuse pleural thickening, but do not meet the criteria set by the ILO classification," i.e., individuals with diffuse pleural thickening and no blunting. Dr. Welch, p.19, suggests that for individuals with DPT and no blunting "in that case these individuals can have an individual review which is based on interpretation of a high resolution CT scan." She suggests that since 83% of the patients in the CARD mortality study meet the requirements of 3mm thickness or over 25% of 25 extent of the chest wall, "it is therefore likely that substantially fewer than 17% of Libby Claimants with diffuse pleural thickening would require individual review." Welch omits to mention that 46% of the CARD patients who died of ARD did not have blunting. Dr. Whitehouse Report 3/14/09, ¶ 75. Almost half are excluded by the TDPs, which call DPT without blunting a "pleural plaque." This is a false result.

Welch discusses alternatives at p.18:

> The alternatives are (1) to accept the statement from any doctor that there is asbestos-related disease on radiograph, or (2) to require all radiographs to be read by a panel of experts. The first option contains no criteria for training or standardization, and the second option would be cumbersome and expensive.

Since the "severe pleural" category is created for Libby, and since most Libby patients have CT scans, the CT scan should be preferred where available. There will be the reading of the hospital radiologist, and the reading of the treating physician. If trust so elects, there may be a third reading by a designated expert. One additional reading is neither cumbersome nor expensive. The same is true for chest x-rays.

7. DLCO.

Welch, p.19, states that for DLCO "there is no agreed upon set of predicted values." Sets of norms vary for all lung function test parameters. ATS/ERS (2005) "Interpretive Strategies," Table 4, indicates no specific set of norms for lung volumes or DLCO. This does not inhibit Dr. Welch from approving TLC (total lung capacity) in the medical criteria. Even though there is no agreed upon set of predicted values, Dr. Welch acknowledges that DLCO is recommended by the ATS (2004) Official Statement as a part of the evaluation of asbestos-related disease. DLCO is also required in the evaluation of permanent impairment in lung disease, in the AMA Guides to the Evaluation of Permanent Impairment, 5th Ed.

Welch, p.19, states: "that DLCO is also decreased in emphysema and therefore a reduction in DLCO is less specific for interstitial lung disease than is a reduction for lung volumes."

Under the medical criteria, all patients who submit claims must have a diagnosis of ARD. In that diagnosis, ATS (2004) Official Statement, p.691, requires that there be "exclusion of alternative plausible causes for the findings." In all cases emphysema has been considered. In most cases emphysema has been ruled out in making the initial diagnosis. Therefore, emphysema is not in the picture. In the minority of cases where there is both ARD and emphysema, a judgment call must be made by the treating physician as to whether the ARD is a significant contributing factor in the low DLCO. There are many judgment calls which must be made by a treating physician. (1) Is there a diagnosis of ARD? (2) Is the interstitial fibrosis asbestos-related or not? (3) How is the CT scan evaluated? (4) What is the extent of diffuse pleural thickening? (5) Can what is seen be called blunting? The presence of a judgment call in a small fraction of cases is no reason to exclude DLCO, which is one of the standard measures for evaluating severity of ARD.

Welch, p.19, goes on to say "a reduced DLCO with a normal TLC and normal FVC could be due to emphysema or some other interstitial lung disease and not asbestosis." A normal TLC certainly does not suggest emphysema. ATS/ERS (2005) Interpretative Strategies, p.955, states: "An increase in TLC, RV, or RV/TLC ratio above the upper limits of natural variability may suggest the presence of emphysema." TLC would generally need to be above 120% of predicted to suggest emphysema. As to "some other interstitial lung disease," that was necessarily ruled out in diagnosing

ARD in the first place. Dr. Welch presents no good reason to depart from standard practice per ATS (2004) Official Statement and the AMA Guides and abandon use of diffusing capacity as a measure of severity.

8. FEV1/FVC ratio.

The medical criteria in the TDPs exclude any patient with an FEV1/FVC ratio under 65. ATS (2004) clearly recognizes that ARD causes obstructive disease. Setting the medical criteria for the FEV1/FVC ratio at 65 will exclude many claimants who have obstructive defect due to ARD.

Welch, p.19, states: "Obstructive disease is defined as an FEV1/FVC ratio of 70%." 70% (actually an absolute number of 0.70) is traditional, but no longer official. ATS/ERS (2005), "Interpretative Strategies" warns against using 0.70 (or 70%):

> The practice using 0.70 as a lower limit of the FEV1/FVC ratio results in a significant number of false positive results in males aged over 40 years and females over 50 years [12], as well as in a risk of over diagnosis of chronic obstructive pulmonary disease (COPD) in asymptomatic elderly never smokers [19].

Markowitz et al (1997) uses a norm of 0.70 for persons under age 60, and 0.65 for persons over age 60. Since most of the Libby patients with significant lung function loss are over 60, 0.65 should be used as norm. The medical criteria for the TDPs use 0.65 as the minimum number to qualify and will exclude a large percentage of patients who have an FEV1/FVC ratio under 0.65, secondary to obstructive defect from asbestos-related disease.

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DATED this 14 day of May, 2009.

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